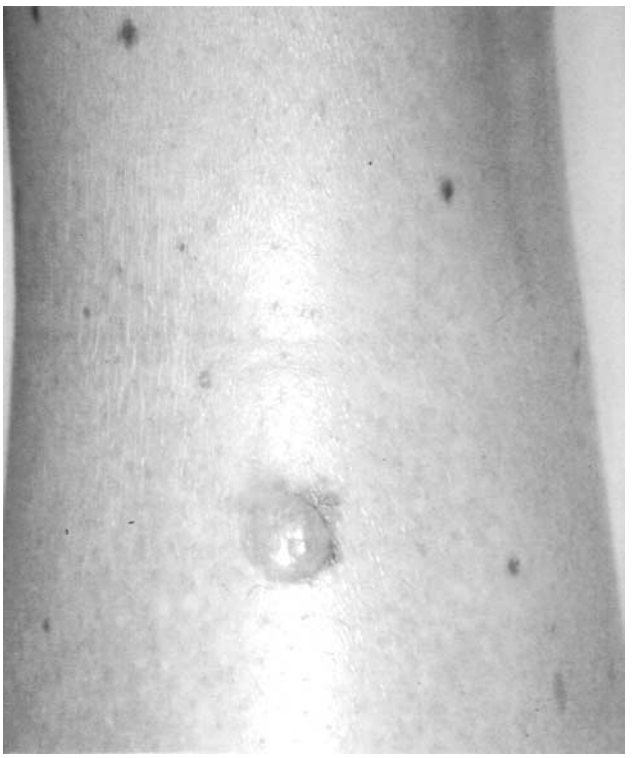

Figure 3 **Lesion in patient 3**

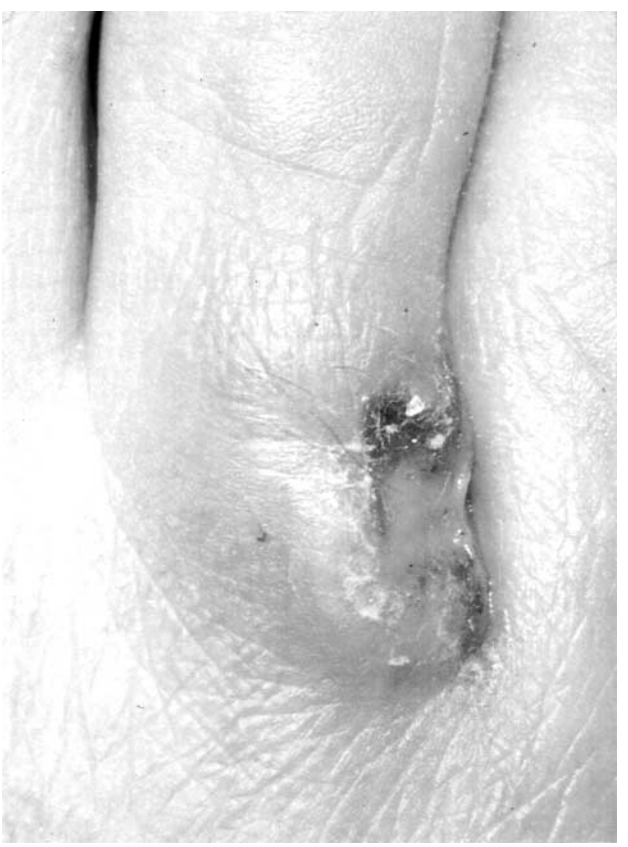

Figure 4 **Lesion in patient 4**

other cutaneous lesions, but even pigmented melanomas are commonly misdiagnosed—especially as melanocytic naevus, basal cell carcinoma, seborrhoeic keratosis or lentigo.[5]

In a study comparing the ability of general practitioners and dermatologists to discriminate pigmented lesions the general practitioners made an exact diagnosis of melanoma in 50% of the cases compared with the dermatologists' 84%.[6] Any changing or atypical mole or non-healing skin lesion should be referred urgently to a dermatologist or to a surgeon with a special interest in pigmented lesions.

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Acute haemolytic anaemia after inhalation of amyl nitrite

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Haemolytic anaemia can be caused by drugs, including some used for recreational purposes.

CASE HISTORY

An Afro-Caribbean man aged 35 was transferred from a psychiatric unit where for six months he had been receiving inpatient care for schizophrenia. For the past two days he had felt generally unwell with fevers. He had passed red urine and his sclerae had become yellow. There was no associated abdominal pain and his stools had been of normal

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colour. Apart from the schizophrenia he had a history of hypercholesterolaemia and sickle-cell trait. His long-term medications were simvastatin and depot flupenthixol. His alcohol consumption was 28 units per week. On examination he was icteric and mildly dehydrated but afebrile. Full blood count on admission showed a normocytic anaemia (haemoglobin 8.2 g/dL, mean corpuscular volume 98 fL), a white cell count of 21.2×10^9/L with neutrophilia and platelets 251×10^9/L. Total bilirubin was 132 μmol/L (normal 0–19) and conjugated bilirubin was 19 μmol/L (0–4). Liver function tests were normal except for an aspartate aminotransferase of 82 iu/L (7–40). Dipstick urinalysis revealed blood and protein. Tests for viral hepatitis were negative. A blood film the following day showed irregularly contracted red cells with puddling, red cell fragments, spherocytes, nucleated red blood cells and hemighosts with 6.9% reticulocytes (0.2–2.0). Direct and indirect Coombs tests were negative and serum haptoglobins were undetectable. The urine contained haemoglobin but not haemosiderin or bilirubin. The results were compatible with non-immune intravascular haemolysis. The sickle-cell trait was confirmed by electrophoresis. His haemoglobin subsequently dropped to 6.4 g/dL, requiring a four-unit blood transfusion, and then remained stable at 13 g/dL.

The patient's admission had been voluntary, and outside the psychiatric unit he had been obtaining cannabis daily and crack cocaine occasionally. In addition, two days before the development of red urine, he had inhaled amyl nitrite ('poppers'). This was the first time he had used poppers for several months, and on previous occasions there had been no similar symptoms. Further investigation, such as measurement of glucose-6-phosphate dehydrogenase (G-6-PD), was impossible because the patient did not attend for follow-up.

COMMENT

We suspect that the severe intravascular haemolysis in this patient resulted from his inhalation of amyl nitrite. A few cases of this sort have been reported previously.[1–3]. The possibility of G-6-PD deficiency cannot be excluded as a contributory factor, although the lack of previous episodes argues against it. Potent oxidants such as cyclic aromatic compounds, chlorate and nitrites can produce haemolytic anaemia in the absence of G-6-PD deficiency by damaging the erythrocyte membrane.[4] Haemoglobin oxidized from the ferrous to the ferric form is known as methaemoglobin (MetHb) and cannot transport oxygen.[5] Acute oxidative damage can lead to the formation of Heinz bodies (precipitated haemoglobin aggregates) which are cleared by the spleen and may not be detected on a peripheral blood film. In addition, oxygen transport is hampered by methaemoglobinaemia, due to oxidation of haemoglobin.[5] Heinz bodies occur after the inhalation of just 30 mL of amyl nitrite,[1] and a high proportion of hemighosts is a marker for poor outcome.[6]

Methaemoglobinaemia can be treated with intravenous methylene blue 2 mg/kg,[2] and this is advised when the concentration exceeds 30–40%. This method depends on intact G-6-PD and phosphate shunt pathways and is therefore ineffective in G-6-PD deficiency. When MetHb levels and the proportion of hemighosts are high, exchange transfusion may be indicated.

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Recurrent axillary vein thrombosis as a manifestation of syringomyelia

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Neuropathic arthropathy of the shoulder can complicate many diseases in adults and children. In the early stages, the manifestations can raise diagnostic challenges.

CASE HISTORY

A man of 53 sought advice when his right arm became grossly swollen. He gave no history of trauma or shoulder

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